The Role of Esophagogastroduodenoscopy Surveillance for Patients with Barrett Esophagus

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KEYWORDS

• Barrett esophagus • Surveillance • Esophagogastroduodenoscopy • GERD
• Esophageal adenocarcinoma

KEY POINTS

- Barrett esophagus (BE) is a precursor to esophageal adenocarcinoma (EAC), but malignant transformation of dysplastic epithelium is rare.
- Patients with BE should be managed with proton pump inhibitors (PPIs) to control symptoms.
- Elderly white men with chronic gastroesophageal reflux disease (GERD) are at highest risk of BE and progression to EAC.
- Patients with BE and evidence of dysplasia on endoscopy should undergo routine surveillance according to the major gastroenterological society guidelines.

INTRODUCTION

GERD affects 40% of the general population at some point in their lives, and up to 20% of US adults report symptoms on a weekly basis.[1–3] Patients with GERD are at risk for barrett esophagus. BE is a metaplastic change in the lining of the esophageal mucosa from its normal squamous epithelium to specialized, columnar intestinal epithelium.[2–5] BE affects 5% to 6% of the average population (approximately 3 million people diagnosed annually in the United States) and up to 25% of the elderly population.[6,7] Although the overall risk for esophageal adenocarcinoma is low (26 cases per 1 million in general US population), its incidence is on the rise in Western populations, with a 300% increase since the 1970s.[4,8] Most patients with EAC present symptomatically at a stage associated with few curative interventions available and a poor prognosis. The 5-year survival rate of EAC is less than 10% and this number has not been affected by current screening and surveillance efforts.[2,8]

BE is diagnosed and monitored through endoscopy, using visual and histologic criteria, and then characterized by length and severity. First, visualization of salmon-colored

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columnar epithelium in the normally white to light pink-colored tubular esophagus suggests proximal displacement of the squamocolumnar junction.[2] Second, biopsy of the tubular esophagus confirms the presence of columnar intestinal epithelium, typically containing goblet cells. If the length of the displaced squamocolumnar junction is longer than 3 cm, it is considered long-segment BE (LSBE) and shorter than 3 cm is considered short-segment BE (SSBE).[4]

EAC is thought to arise from BE through progressive dysplasia. This association raises concern for the need for screening and surveillance of patients with chronic GERD and BE to identify those at risk for progression to EAC. Although at first glance this seems logical, the utility, efficacy, and cost of such an approach must be considered. Patients with BE have a decreased quality of life compared with the general population due to inappropriately high level of fear for their risk of progression to cancer, unnecessary testing and visits, and increased life insurance premiums. In 1 study, patients diagnosed with BE experienced a 100% increase in life-insurance premiums compared with their age-matched and gender-matched controls, and some were unable to obtain insurance at all after diagnosis with BE.[9,10]

DEVELOPMENT OF BARRETT ESOPHAGUS AND ESOPHAGEAL ADENOCARCINOMA

GERD is thought to progress to BE through frequent, severe, and/or long-term exposure to acid and bile reflux. This reflux alters the normal squamous epithelium of the esophagus, which is replaced with columnar epithelium.[1,2,5] Although BE is a precursor to EAC, most patients with BE do not progress past nondysplastic or low-dysplastic disease.[2,11] The yearly esophageal cancer risk for patients with GERD who are 50 years or older is only 0.04%, and the risk of progression from BE to EAC is only 0.5% per patient year.[2,4,6]

Risk factors for the development and progression of BE and EAC include onset of symptoms before age 30, history of severe GERD symptoms, and greater than 20 cumulative years of exposure (**Fig. 1**).[1,4] The use of the term, *alarm symptoms*, describes the symptoms a patient may experience as this progression occurs. These alarm symptoms include dysphagia, anemia, weight loss, bleeding, and recurrent vomiting.

Chronic acid exposure increases the risk for BE and the subsequent segment length and severity of BE. For reasons that are not understood, men are in the highest risk group, accounting for 80% of cases of EAC. To give some perspective, rates of EAC in women are equivalent to the prevalence of breast cancer diagnoses in men.[3] In particular, elderly white men seem at the highest risk. Elderly patients are at high risk due to increased time for symptom exposure as well as a high frequency of atypical symptoms, thought to be due to less sensation to the typical heartburn symptoms of reflux.[12] Obesity is associated with increased GERD symptoms and erosive esophagitis, due to the increased risk of reflux and hiatal hernia in patients with an elevated body mass index (BMI). Patients with central obesity in particular are at an increased risk of BE due to elevated intragastric pressure.[2–4] Alcohol use and smoking have also been suggested risk factors for development of esophageal adenocarcinoma, but more recent data have been conflicting.[1,2,6,13,14]

In summary, elderly white men with chronic GERD symptoms, hiatal hernia, and elevated BMI are at greatest risk for developing severe BE and progression to EAC. Although having ever used a PPI is thought to be a risk factor for BE, one group excluded from this risk are those on a PPI due to history of *Helicobacter pylori*. Several studies have found a protective effect of prior *H pylori* infection and BE, which is not thought to be linked solely to adequate treatment of *H pylori* infection.[1,2]